U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 30 April 2004

Case No. 2003-BLA-47

In the Matter of

EDITH K. WILTROUT
SURVIVOR OF WALTER WILTROUT
Claimant

v.

SHANNOPIN MINING COMPANY
Employer

and

OLD REPUBLIC INSURANCE COMPANY
Carrier

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

Robert F. Cohen, Esquire
For the Claimant

Gregory J. Fischer, Esquire
For the Employer

Before: MICHAEL P. LESNIAK
Administrative Law Judge

DECISION AND ORDER—AWARDING BENEFITS

and

ORDER GRANTING CLAIMANT'S MOTION FOR PAYMENT OF DEPOSITION EXPENSES

This case arises from a claim for benefits under the "Black Lung Benefits Act," Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. § 901 *et seq*. (hereinafter referred to as "the Act"), and applicable federal regulations, mainly 20 C.F.R. Parts 412, 718, and 727 ("Regulations").

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis or to the survivors of persons whose death was caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as black lung.[1]

At a formal hearing conducted in Pittsburgh, Pennsylvania on June 25, 2003, all parties were afforded a full opportunity to present evidence and argument, as provided in the Act and Regulations issued thereunder.[2] The record was held open to submit a written stipulation of the evidence, closing briefs, for Claimant to file any motions pertaining to cost issues and applicability of regulations, and for Employer to file a response to Claimant's motions, if any. Tr. 48.

The parties filed a joint "Stipulation of Evidence" on October 30, 2003. Employer filed its closing comments on December 2, 2003 and Claimant filed her closing brief on December 9, 2003. Rulings on the evidentiary objections, raised at and subsequent to the hearing, are resolved in this Decision and Order.

ISSUES[3]

The contested issues are:

1. Modification based on mistake of fact;
2. Whether the miner's coal mine dust exposure contributed to or hastened his death.

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[1] The following abbreviations are used in this decision: DX = Director's exhibit; EX = Employer's exhibit; CX = Claimant's exhibit; JX = the parties' joint stipulation of evidence; Tr. = Transcript of the hearing.

[2] At the hearing, the Director's exhibits 1–28, 30–34, 38–42, 44, 46–61, and Claimant's exhibits 1–8 were admitted into evidence without objection. Tr. 6–10. Claimant objected to portions of Employer's exhibit 4, which is resolved *infra*. In addition, Employer's exhibits were never officially admitted into evidence at the hearing. Tr. 12. Accordingly, I hereby admit Employer's exhibits 1–7 into the record.

[3] Employer stipulated at the hearing that the miner had pneumoconiosis that arose out of his coal mine employment. Tr. 33.

PROCEDURAL HISTORY AND FACTUAL BACKGROUND[4]

Procedural History

Claimant Edith K. Wiltrout filed her claim for benefits on June 30, 1997. It was denied by the District Director on February 4, 1998. DX-2 at 25. Claimant requested and received a formal hearing before Administrative Law Judge Daniel L. Leland, who awarded benefits by Decision and Order on January 5, 1999. DX-32. Employer appealed Judge Leland's decision to the Benefits Review Board, which issued a Decision and Order vacating Judge Leland's decision and remanding the case for further proceedings. DX-33, -39. On May 11, 2000, Judge Leland issued a Decision and Order on Remand—Denying Benefits. DX-40. Claimant appealed Judge Leland's Decision and Order on Remand to the Benefits Review Board, which affirmed the denial of benefits on May 31, 2001. DX-46. Claimant then requested modification of the denial of benefits on October 31, 2001. DX-47. The District Director denied the request for modification on July 18, 2002. DX-55. Claimant disagreed with the determination and requested a formal hearing on August 9, 2002. DX-56. The case was referred to the Office of Administrative Law Judges on November 7, 2002 for hearing. DX-59, -60, -61. A hearing was held on June 25, 2003 in Pittsburgh, Pennsylvania.

The findings of fact and conclusions of law that follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and the testimony presented. Where pertinent, I have made credibility determinations concerning the evidence.

Background

At the hearing, Claimant testified as follows: Claimant is the widow of the miner, Walter Wiltrout, who died on June 4, 1997 at the age of 67. Tr. 35, 40. Claimant stated that she has not remarried since her husband died. *Id*. Claimant testified that her husband began smoking in 1948—although she did not know him at that time—and he stopped smoking in 1960. *Id*. Claimant further testified that she considered her husband a light smoker because he was not allowed to smoke in the mines, where he worked for eight to ten hours a day. She estimated his smoking to be about a pack per day. Tr. 36, 45. Claimant stated that her husband decided to stop smoking after the initial reports about the dangers of smoking came out. Tr. 36. Claimant testified that she does not remember what brand of cigarettes her husband smoked, nor does she recall whether they were filtered or unfiltered. Tr. 45–46.

Claimant testified that her husband had shortness of breath, which she first noticed in July 1994, when they were on a trip to Pike's Peak. Tr. 37. She explained that they took a train ride to the top and that he could not breathe at the high altitude. *Id*. Claimant stated that she reported the incident to her husband's doctor, who subsequently prescribed medications for him to take two days prior to other trips out west. *Id*. Claimant testified that her husband also went

[4] Given the filing date of this claim, subsequent to the effective date of the permanent criteria of Part 718 (*i.e.*, March 31, 1980), the regulations set forth at 20 C.F.R. Part 718 will govern its adjudication. Because the miner's last exposure to coal mine dust occurred in Pennsylvania, this claim arises under the jurisdiction of the U.S. Court of Appeals for the Third Circuit. *See Broyles v. Director, OWCP*, 143 F.3d 1348, 21 BLR 2-369 (10th Cir. 1998).

to Colorado to hunt and fish, and that other family members told her that he had difficulty breathing at that time, too. Tr. 38. She testified that the miner did not go on any hunting trips out west before or after his trip to Colorado in 1995. Tr. 38, 46. Claimant further testified that her husband did hunt locally until the year he died, but he did not have to do much walking or cover hilly terrain. Tr. 46. Claimant stated that although her husband caught deer he did not have to carry them. Tr. 47.

Claimant stated that she believes her husband's breathing problems worsened after 1994 because he frequently complained of being tired after working a little bit at their summer home. Tr. 39. Claimant explained that after working in the garden or stacking wood, her husband would complain of being tired and he would lie down and rest but she never heard him gasping for air. Tr. 43, 48.

Claimant testified that her husband last worked at Shannopin Mining Company in 1987 and he left due to a severe shoulder injury for which he received worker's compensation benefits until the time he died. Tr. 40. Claimant further testified that the miner's last coal mine job was as a mine foreman and that he did not work in any type of employment after 1987. *Id*.

Claimant testified that her husband was never hospitalized for his breathing problems. Tr. 41. She also stated that his breathing difficulties were most noticeable at night, when he slept with two pillows, and when he laughed hard she would hear him wheezing. Tr. 37, 41. Claimant testified that her husband's primary physician was Dr. Elnicki, who also treated him for non-insulin-dependent diabetes, which the miner controlled with diet. Tr. 42. Claimant testified that her husband did not have heart problems during his lifetime. Tr. 43. Claimant testified that she was with her husband on the day he died. Tr. 43–44. She stated that he experienced indigestion and difficulty breathing on the day he died, but did not complain of any pain. Tr. 44.

MEDICAL EVIDENCE

Previously Submitted Evidence

Except as modified or superseded herein, the medical opinion evidence set forth in Judge Leland's Decision and Order—Awarding Benefits and Decision and his Order on Remand—Denying Benefits, is incorporated by reference herein. I find that Judge Leland reviewed the evidence before him accurately. Such evidence is also summarized in the parties' joint stipulation of evidence and closing arguments and I note that neither party finds fault with Judge Leland's findings, given the medical evidence before him at the time. Indeed, Claimant's attorney states:

> It is not the claimant's contention that Judge Leland's Decision and Order on Remand was irrational given the medical evidence before him. Judge Leland rationally concluded that Dr. Kleinerman's qualifications were superior to those of Dr. Wecht and Dr. Oesterling, that Dr. Wecht's analysis was simplistic, that the analysis by Drs. Oesterling and Kleinerman was more thorough than the analysis by Dr. Wecht, that Dr. Elnicki's credentials were not included in the record, and

> that Dr. Elnicki's opinion was speculative …. These conclusions were reasonable given the evidence before Judge Leland …. But the evidence now is very different. With the opinions of Dr. Green and Dr. Abraham, together with the new opinion by Dr. Elnicki, claimant has established that the ultimate conclusion reached by Judge Leland was incorrect.[5]

The new medical evidence submitted in connection with Claimant's request for modification is summarized below.

Medical Reports and Depositions

Dr. Cyril H. Wecht

The deposition of Dr. Wecht was taken on August 4, 1998 at appears in the record at EX-5. Dr. Wecht testified that he is board certified in anatomic, clinical, and forensic pathology and holds six faculty positions at the University of Pittsburgh and Duquesne University, including the University of Pittsburgh Schools of Medicine, Dentistry, and Public Health and Duquesne University Schools of Law, Pharmacy, and graduate school of Health Sciences. Dr. Wecht testified that the disease process of coal workers' pneumoconiosis can progress in the absence of further exposure to coal dust and the clinical signs and symptoms may worsen because the disease continues to wreak havoc with the pulmonary system and, in some instances, the cardiovascular system. EX-5 at 10–12. Dr. Wecht stated that he was told the miner smoked for 12 to 15 years but he does not consider it to be a significant smoking history because he stopped 34 years before his death and one doesn't get problems starting after all those years. EX-5 at 15.

Dr. Wecht testified that on autopsy the miner had hypertensive and arteriosclerotic cardiovascular disease, no evidence of myocardial infarction, enlarged heart with moderate atherosclerosis of the coronary arteries and chronic obstructive pulmonary disease. EX-5 at 16. Dr. Wecht described the miner's coal workers' pneumoconiosis as moderate—a moderate degree of simple coal workers' pneumoconiosis. EX-5 at 17. Dr. Wecht testified that if one has macules less than one millimeter, he thinks one would not have clinical problems. EX-5 at 30. Dr. Wecht testified that in the term "simple" pneumoconiosis, the word simple us not to be construed as synonomous with mild. EX-3 at 33. Dr. Wecht testified that the miner had moderate emphysema, based on the blebs and bullae ranging from 2.5 – 4 centimeters and 1 – 1¾ inches, and he had cardiomegaly. EX-3 at 43. He stated that both sides of the heart were enlarged, but the right side much more so. EX-3 at 44. Dr. Wecht testified that absent any preexisting lung disease, the miner's hypertensive and coronary arteriosclerosis was sufficient to have caused his death. EX-3 at 46. Dr. Wecht stated that his finding of passive congestion of the lung is a complication of a failing left side of the heart or a secondary problem caused by a failing heart. EX-3 at 48.

Dr. Francis H. Y. Green

The medical report of Dr. Green is dated January 29, 2002 and appears in the record at DX-54 and -51. Dr. Green, board certified in anatomic pathology, is a professor in the

[5] *See* Claimant's closing brief at pp. 19–21.

Department of Pathology at the University of Calgary Medical Center in Alberta, Canada. DX-51. Dr. Green reviewed the miner's records at Claimant's request. The records Dr. Green reviewed consisted of the autopsy report, death certificate, autopsy slides, miner's medical records, consultation reports of Drs. Oesterling, Tuteur, and Kleinerman, and the depositions of Drs. Wecht and Oesterling. Dr. Green's diagnoses are: *Heart*—severe right ventricular hypertrophy (based on Dr. Wecht's autopsy report); moderate left ventricular hypertrophy; subendocardial fibrosis. *Lungs*—mild, simple macular coal workers' pneumoconiosis; centriacinar emphysema; severe pulmonary hypertensive changes; acute aspiration. *Other organs*—benign prostatic enlargement; mild fatty change of liver; acute and chronic congestion of liver and spleen; pleural plaque.

Dr. Green opined that the immediate cause of death appeared to be a cardiac event leading to ventricular fibrillation. He noted that the autopsy ruled out other causes of sudden death and that although sudden cardiac death is often caused by ischemic heart disease, the autopsy failed to show significant atherosclerotic disease of the coronary arteries. He noted that while the miner's vessels were diseased, they were within the normal range for a man the miner's age and that this severity of disease is not associated with sudden death. Dr. Green agreed with Dr. Kleinerman that there was no evidence the miner suffered from hypertension at the time of death. Dr. Green noted that the abnormality of the heart was right ventricular hypertrophy, with the degree of the hypertrophy not accounted for by the mild left ventricular hypertrophy that was also present. He explained that while left ventricular hypertrophy can cause small increases in size of the right ventricle, in the miner's case there was no evidence that left ventricular disease caused the right ventricular hypertrophy because there was no history of the left ventricular failure necessary to affect right ventricular function. Dr. Green further explained that the right ventricular changes seen were out of proportion to the disease in the left ventricle. Therefore, he concluded that the miner had anatomic evidence of cor pulmonale and the liver and spleen showed acute and chronic venous congestion, which are classic features of right ventricular failure in cor pulmonale.

Dr. Green noted that the miner had evidence of coal workers' pneumoconiosis and emphysema, both of which can cause cor pulmonale. He explained that cor pulmonale is often the cause of death in patients with emphysema, with more severe emphysema being more likely to develop into cor pulmonale. Dr. Green stated that severity of emphysema can only be diagnosed on macroscopic examination of the whole lungs because emphysema results in large empty airspaces and cannot be sampled on slides. He noted that Dr. Wecht described numerous emphysematous blebs and bullae in the upper lobes, which Dr. Green opined constitutes moderately severe emphysema. Dr. Green also noted severe arterial narrowing, including muscularization of the small arterioles and plexiform lesions; the latter, he explained, only develop in severe pulmonary hypertension. Dr. Green explained that pulmonary hypertension is not associated with abnormalities on conventional pulmonary function testing but it would lead to breathlessness on exertion and at high altitudes, such as that experienced by the miner. Dr. Green noted that, on autopsy, Dr. Wecht found pulmonary osteoarthropathy, or clubbing of the fingers, which is commonly associated with long-standing and severe lung disease. Dr. Green concluded that the miner suffered from cor pulmonale secondary to coal workers' pneumoconiosis and emphysema and he further opined that cor pulmonale, as opposed to left heart disease, led to the sudden arrhythmia and death.

Dr. Green opined that the mild simple coal workers' pneumoconiosis found on autopsy would have resulted from the miner's exposure to coal mine dust. He explained that the amount of visible dust on autopsy would have been lessened by clearing of the lungs in the seventeen years preceding the miner's death after he stopped working in the mines. In addition, Dr. Green stated that the absence of pneumoconiosis on x-ray was not surprising as the macules were very small and well below the resolution of the standard x-ray. Dr. Green also strongly disagreed with Dr. Oesterling's opinion that coal dust macules need to be 1 mm or more in dimension to diagnose coal workers' pneumoconiosis. He noted that Dr. Oesterling quoted the College of American Pathologists expert committee report in support of that opinion and that both he and Dr. Kleinerman were on that committee and never specified a minimum size for the coal dust macule. Dr. Green noted that the miner had evidence of moderately severe emphysema but that the miner's brief interlude of smoking, which stopped 37 years before his death, would not have contributed to the emphysema. He stated that the more likely cause for the emphysema was exposure to dust and fumes in the coal mines and he attached a list of references showing that chronic obstructive lung disease, including emphysema, can be caused by coal dust exposure.

Dr. Green opined that the miner died due to a cardiac arrest secondary to heart disease, the most important being cor pulmonale, which resulted from chronic lung disease. He explained that the chronic lung disease constituted simple macular coal workers' pneumoconiosis of mild severity as well as moderately severe emphysema, both of which resulted from coal mine dust exposure. Dr. Green noted that the miner also had mild left ventricular hypertrophy, probably resulting from his previous history of hypertension and which would also have contributed to his cardiac arrest. It was Dr. Green's opinion that pneumoconiosis, including emphysema, was a direct contributing factor of death.

Dr. Green wrote another report dated May 15, 2003 following his review of Dr. Naeye's medical report and deposition. This report appears in the record at CX 5. Dr. Green noted that Dr. Naeye diagnosed minimal simple coal workers' pneumoconiosis, mild emphysema, and small fibrotic foci in a piece of tissue from the left ventricle of the heart and that Dr. Naeye stated that this would be a possible site of origin of the fatal arrhythmia. Dr. Green stated that Dr. Naeye's opinion is notable in that it makes no mention of the state of the pulmonary vessels in the miner's lung, which he considers surprising considering Dr. Wecht diagnosed cor pulmonale on autopsy. Dr. Green noted that Dr. Naeye diagnosed mild centrilobular emphysema, and stated that an exception to Dr. Naeye's testimony that pathologists take tissue samples from the most diseased part of the lungs, occurs with the diagnosis of emphysema. Dr. Green explained that with emphysema, it would be impossible to sample the most severe lesions because "one would be sampling thin air." Dr. Green further explained that emphysema is caused by the destruction of lung tissue leaving large empty spaces which, when greater than 1 centimeter, are called bullae. He stated that this is why most pathologists and text books agree that the only way to reliably diagnose the severity of emphysema is by examination of the cut surface of the whole lung. Dr. Green noted that Dr. Wecht described the presence of bullae in the miner's lung and stated that his own evaluation of the miner's emphysema as being of moderate severity was based primarily on Dr. Wecht's gross description of the lungs supplemented by the microscopic evidence of emphysema.

Dr. Green stated that he disagrees with Dr. Naeye's opinion that the effect of coal mine dust on emphysema is mild, as several epidemiologic and pathologic studies attempt to quantify the relative contribution of coal mine dust exposure to declines in FEV1 (surrogate of emphysema) or to pathologic grade of emphysema. Dr. Green stated that data indicates that one year of average smoking contributes approximately the same degree to the emphysema score as one year of underground mining. He explained that the mechanism for producing emphysema by coal mine dust and cigarette smoke is identical and the effects are not distributed equally in the population, so some individuals are much more susceptible to the harmful effects of cigarette smoke and coal dust. Dr. Green stated that his opinion is that there is now a consensus of scientific opinion that coal mine dust exposure can, on its own, produce disabling emphysema. Dr. Green attached a list of references relevant to this opinion.

Dr. Green attached an article to support his opinion that persons quitting smoking before age 40 are unlikely to develop severe emphysema, and agreed with Dr. Naeye's statement that there is a lot of variability in the degree of emphysema based on genetic and familial factors. Dr. Green stated that when he encounters significant emphysema in a person under 50, he looks for a genetic predisposition. Dr. Green noted that Dr. Naeye disagreed with his opinion that macules may break down in the presence of emphysema, making recognition of the disease more difficult. He cited an article he co-wrote in *Human Pathology* that demonstrated that as emphysema in coal miners increased in severity, the black pigment was dispersed into the bloodstream and taken up by the liver and spleen. Dr. Green discussed Dr. Naeye's opinion that there is an excess of sudden death in cigarette smokers related to the destruction of normal muscle fibers in the arterioles of the heart. He explained that Dr. Naeye did not describe this lesion in the miner's heart and there is no mention of these specific lesions in the arterioles that he is describing. In addition, Dr. Green noted that Dr. Naeye's report did not describe the lesions of the microcirculation that he discussed in his deposition, nor did Dr. Green observe them himself.

Dr. Green noted that Dr. Naeye doubted the diagnosis of cor pulmonale made by Dr. Wecht because he stated that for many years Dr. Wecht did not measure the thickness of the ventricles from the base of the trabeculae. Dr. Green explained that Dr. Wecht addressed this issue in his report and that Dr. Wecht's description of measuring the heart is the current accepted practice for measuring the thickness of ventricles and can be found in standard autopsy practice textbooks. In addition, he explained that Dr. Wecht's method is also the required method for submission of cases to the National Coal Workers' Autopsy Study. Dr. Green stated that he has no doubt Dr. Wecht made the appropriate measurements and that they indicate that the miner had cor pulmonale.

Dr. Green was deposed on January 8, 2003. EX-3, CX-4.[6] He testified that he is board certified in anatomic pathology, is a full professor of pathology and head of the autopsy service, divides his time equally between patient care and university duties including teaching, research, and administration, and was chair of the respiratory research group from 1996–2002.

[6] The deposition transcript of Dr. Green contained grammatical/typographical errors that Dr. Green subsequently corrected. Claimant's counsel submitted the list of corrections as CX-4. Thus, the deposition with the corrections appears as both EX-3 (deposition transcript) and CX-4 (corrections). I incorporated the corrections and cite to the deposition as EX-3, hereinafter.

In addition, Dr. Green stated that he was chief of the pathology section at NIOSH and in charge of the National Coal Workers' Autopsy study. EX-3 at 4–7. Dr. Green testified that he has a particular interest in the relationship of pulmonary emphysema and coal dust exposure. In his NIOSH research, he showed a relationship between the amount of dust in the lung and the amount of emphysema and a relationship between the severity of emphysema with the severity of pneumoconiosis. EX-3 at 8–9. Dr. Green testified that he continues to perform research in this area with NIOSH on the National Coal Workers' Autopsy Study, which now includes about 8,000 cases that have now been accessioned. EX-3 at 11.

Dr. Green testified that the miner died a sudden death after experiencing ventricular fibrillation. EX-3 at 13. He stated that factors he considered important in relation to the death are a history of minimal heart disease confined to hypertension, diabetes, and lung disease. EX-3 at 14. Dr. Green stated that the miner experienced breathlessness at high altitude, under exertion, and had chronic productive sputum. EX-3 at 15. Dr. Green stated that the miner's pulmonary function studies were normal. *Id*. Dr. Green stated that the miner had 34 years of underground coal mine employment and that he smoked one pack per day for 13 years, stopping at age 30. EX-3 at 16. Dr. Green stated that he based his diagnosis of right ventricular hypertrophy on Dr. Wecht's measurements of the left and right ventricles; the right ventricular measurement was double the normal size. EX-3 at 17. Dr. Green stated that his second diagnosis was left ventricular hypertrophy based on the 25 percent increase in the size of the left ventricle, and it is the amount of a person with mild hypertension. EX-3 at 20. Dr. Green testified that his third diagnosis was subendocardial fibrosis, which he explained is scar tissue in the heart muscle, on the inside of the heart, so it would not be consistent with an old myocardial infarct. EX-3 at 20–21.

Dr. Green testified that, with regard to the lungs, his first diagnosis was mild simple macular coal workers' penumoconiosis, because in the slides there were lesions of simple CWP and the miner had both macules and emphysema around them called "focal emphysema." EX-3 at 21–22. Dr. Green's response to Dr. Oesterling's criticism that the macular lesions were too small to be pneumoconiosis is that there is no minimum size criteria for the definition of a macule. EX-3 at 22–24. Dr. Green testified that his second lung diagnosis was centriacinar or centrilobular emphysema. EX-3 at 24. Dr. Green explained that he relies more on the prosector's gross description of the lung in classifying emphysema because sampling severe emphysema is difficult, as there is often nothing on the slide. EX-3 at 25. Dr. Green stated that Dr. Wecht described bullae and blebs two to four centimeters across, which is quite large. *Id*.

Dr. Green testified that his third lung diagnosis was severe pulmonary hypertensive changes. *Id*. He explained that these changes occur when the pressure goes up, the walls thicken and in severe cases, plexiform lesions develop, which are small aneurysms that were present in the miner. EX-3 at 26. He stated that most experts in lung pathology consider this the most advanced form of pulmonary hypertension. *Id*. Dr. Green distinguished pulmonary hypertension, which involves the right heart and lungs, from systemic hypertension, which involves the left ventricle. *Id*. Dr. Green stated that Dr. Perper essentially described the same finding in his report but he did not see any findings of pulmonary hypertension in the reports of Drs. Kleinerman and Oesterling, and neither of them commented on the blood vessels of the lung at all. In addition, he noted that although Dr. Kleinerman opined that cor pulmonale did not

exist, the vessels would be the best way to determine whether the miner had pulmonary hypertension or cor pulmonale, and he did not address them. EX-3 at 26.

Dr. Green stated that his fourth lung diagnosis, acute aspiration, is not significant other than that it fits with sudden death, because people aspirate when they have cardiac arrest and aspiration may occur during the resuscitation process during intubation. EX-3 at 29. Dr. Green stated that the most important diagnosis for the other organs is the acute and chronic congestion of the liver and spleen. *Id*. He stated that the spleen was almost three times normal size and the liver was 50 percent bigger, with both of them showing evidence of chronic venous congestion. Taken together, these are classic signs of right-sided heart failure. EX-3 at 30. Dr. Green explained that the miner did have some ischemic heart disease, but that the amount the miner had was not out of the ordinary for someone his age and was not at the level associated with sudden death. EX-3 at 32. Dr. Green stated that he disagrees with Dr. Oersterling's opinion that the cause of the miner's cardiac arrest was ischemic heart disease because there is no evidence of it. EX-3 at 33. He explained that one would need evidence of severe or significant coronary artery disease or an old, acute myocardial infarction or a history of angina or heart disease, none of which the miner had. *Id*. Dr. Green stated that the myocardial fibrosis addressed by Dr. Oesterling may be due to his hypertension, although his blood pressure was normal for years before death, and diabetes can cause some fibrosis in the left ventricle but there was nothing specific for any of those. EX-3 at 34. Dr. Green stated that his view is that there were minor changes in the left ventricle which probably did not cause the miner's death; death resulted from the changes to the right side of the heart which were more severe and much more explainable in terms of the miner's lungs. EX-3 at 36.

Dr. Green stated that he diagnosed cor pulmonale because the miner had anatomic evidence of the disease that included right heart hypertropy, a doubling of the size of the heart, right heart failure evidenced by congestion of the liver and spleen, thickening of the arteries in the lung, evidence of significant lung disease, and evidence of clubbing of the fingers, known as pulmonary osteoarthropathy. EX-3 at 37. He stated that the autopsy report described an increase in the anterior and posterior diameter of the chest, which is a classic sign of emphysema. EX-3 at 38. He stated that there was description on autopsy of moderately severe emphysema and evidence of pneumoconiosis and that, putting everything together, there was evidence of chronic lung disease that caused the pulmonary hypertension, which caused the right heart hypertrophy and eventually the failure that led to a fatal arrhythmia. EX-38, -39. Dr. Green stated that he has seen cases of persons with severe pulmonary impairment and a normal FEV-1. EX-3 at 39. He explained that FEV-1 is not affected by pulmonary hypertension or cor pulmonale at all. *Id*. Dr. Green disagreed with Dr. Kleinerman, who said if the left ventricle is enlarged, one cannot diagnose cor pulmonale because there would need to be failure of the left ventricle, which did not occur here, and there was no evidence of pulmonary edema, which would have arisen from left ventricular failure. EX-3 at 42. Dr. Green explained that in this case, the right heart disease was twice the normal thickness and the left 25 percent, so it does not make sense to ascribe the right heart disease to the minimal left disease that was present. EX-3 at 42–43.

Dr. Green testified that the cause of the miner's cor pulmonale was primarily his emphysema and a minor contributing factor would have been his pneumoconiosis. EX-3 at 43. He stated that the cause of the emphysema was primarily dust exposure and if smoking contributed at all, it might have contributed one-third, but he doesn't believe smoking was

significant because the miner quit at age 30 and studies show that if a person quits before age 40, it has virtually no effect. EX-3 at 44–45. Dr. Green disagreed with Dr. Perper's statement that the minimal, almost nonexistent amount of coal workers' pneumoconiosis could result in any degree of centrilobular emphysema, because the miner's emphysema was related to his cigarette smoking. EX-3 at 48. He explained that he did not think the amount of pneumoconiosis was important because the miner had 34 years of exposure to coal dust, which was more than enough to cause emphysema. *Id*. Dr. Green also disagreed with Dr. Perper's statement that the emphysema was caused by cigarette smoking by reiterating that quitting smoking before age 40 virtually has no effect on subsequent lung function and cited a previously attached article to his medical report. In addition, he stated that even if one assumed smoking contributed, it would have played a minor role, since the miner had only 13 years of smoking and 34 years in the mines. EX-3 at 49.

Dr. Green stated that he identified a very mild form of coal workers' pneumoconiosis that was not complicated or progressive massive fibrosis. EX-3 at 53. He stated that he did not measure any of the macules he saw nor did he record how many he saw. *Id*. Dr. Green noted that Drs. Kleinerman and Oesterling observed only two dust macules in the lung tissue but suspects that is an underestimate because, he explained, when one gets centrilobular emphysema the macules begin to break down and become harder to recognize, and the remaining ones tend to be the rarity. EX-3 at 54. He stated that the CWP was mild but that he suspects it was more severe, based on Dr. Wecht's gross description of the lungs. EX-3 at 55. When asked whether lesions of under one millimeter were indicative of the beginning stage of pneumoconiosis and his response to Dr. Oesterling's supplemental report, Dr. Green stated that the one to four millimeters he discussed in his article referred to whole lung sections and does not refer to microscopic sections or microscopic diagnosis. EX-3 at 58–62. He also explained that there is not a clear-cut relationship of size of a lesion and the severity of disease because if the macules cause narrowing or disturbance of the flow through the small airway, they are going to have a big functional effect which will not be dependent on their size, but on what they are doing in that unit. EX-3 at 62–63. He further explained that with emphysema comes a breakdown of lung tissue and the dust gets redistributed, making it difficult to know what the original size was. EX-3 at 63. Dr. Green stated that the macules would not appear on x-ray and that black pigment in the lung is not equivalent to pneumoconiosis. EX-3 at 64.

Dr. Green testified that the FEV-1 and arterial blood gas studies were normal, increased with exercise, and did not reveal a hypoxemic condition. EX-3 at 67. When asked whether he would expect to see major degradation in the pulmonary function and arterial blood gas studies 14 years after leaving the mines, Dr. Green testified that there are lots of discrepancies in individual cases and that he has seen cases in which the FEV-1 is reasonably well-maintained and the patient has severe emphysema, and also that pulmonary hypertension has no effect on the FEV-1. EX-3 at 70. He stated that there are a lot of studies that show that the relationship between FEV-1 and emphysema is not quite as definite and that the problem with the miner's history is that there is only one measurement at one point in time. EX-3 at 80. He stated that there is no record of what the miner's lungs were like 20 years earlier and that he might have had a super-normal pulmonary test. *Id*. Dr. Green stated that the macular disease seen in the miner's case was insignificant from a clinical perspective of conventional pulmonary testing, and it was not impacting on his ability to oxygenate blood. EX-3 at 71. Dr. Green acknowledged that Dr.

Elnicki's records indicate that the miner smoked for 21 years, from 1947 to 1968, and that there are a variety of smoking histories in the clinical records. EX-3 at 75–76. Dr. Green also acknowledged that he did not consider a smoking history of 31½ pack years and that such an amount might cause a moderate degree of emphysema but that it would depend on the age of the miner when he quit, and he holds that the miner's early quitting is the most significant factor, although the greater his exposure to cigarette smoke was, the greater the risk that it would contribute to underlying lung disease. EX-3 at 76–77. Dr. Green acknowledged that although the lungs are fully developed in the late teenage years, cigarette smoking would possibly have a small effect and more than in an adult. EX-3 at 79.

Dr. Green testified that he noted emphysema in the areas involved with coal dust macules, which would be focal emphysema. EX-3 at 81–82. He acknowledged that the "Archives of Pathology" indicates that focal emphysema is generally not impairing and that Dr. Kleinerman held that opinion, but he thinks there is now no good data showing that focal and centriacinar emphysema are different. He noted that Dr. Kleinerman acknowledged that centriacinar emphysema causes severe impairment. EX-3 at 82. Dr. Green testified that there is a linear relationship between the amount of dust in the lung tissue and the degree of emphysema and that the severity of emphysema is correlated to the severity of the coal workers' pneumoconiosis. EX-3 at 85. In relation to this case, Dr. Green reiterated the mechanism by which the emphysema destroys the lung tissue and the pneumoconiosis is destroyed so the chest x-ray is often negative for pneumoconiosis due to the effect of the emphysema. EX-3 at 86. Dr. Green acknowledged that the majority of the pathologists diagnosed minimal amounts of dust in the lung. EX-3 at 86–87. He explained that dust can be in other areas other than the macules or nodules, it is distributed throughout the lung and, in his opinion, there was dust in moderate amounts in other areas of the lung although he does not know how much they would contribute to emphysema. EX-3 at 87. Dr. Green acknowledged that despite the level of emphysema he diagnosed, the pulmonary function studies were normal. EX-3 at 90.

Dr. Green testified that it is not possible to determine the severity of emphysema based upon the lung disease tissue in slides and that he relied on Dr. Wecht's description to determine the severity of the emphysema. EX-3 at 90–91. He stated that even if Dr. Wecht described the emphysema as moderate, then that would still be enough to cause cor pulmonale; he based his opinion of severe emphysema on the description and measurements in Dr. Wecht's report. EX-3 at 93. He explained that bullous emphysema up to four centimeters is usually advanced emphysema. *Id*. Dr. Green stated that he did not see any notation of clubbing of the fingers or increased AP diameter of the chest in the miner's clinical records. EX-3 at 93–94. He explained that increased AP diameter of the chest is not easy to see and is more obvious when someone is flat on his back on an autopsy table but that clubbing of the fingers should be fairly obvious during a patient's life. EX-3 at 94. Dr. Green testified that the clubbing of the fingernails could not have occurred as a result of the miner's 25 minutes without a pulse before death because it takes many years to develop. EX-3 at 96–97. He testified that the increased AP diameter should not have occurred during resuscitation because once the tube was removed the lungs would fall into their natural position. EX-3 at 97.

Dr. Green stated that he thinks the miner did not have arteriosclerotic heart disease and it is his opinion that the fibrosis in the left ventricle could not account for the changes in the

coronary arteries—although he cannot entirely rule it out. *Id*. He stated about the pathology in the left ventricle that there are a number of causes, one of which is hypertension and another diabetes. EX-3 at 98. Dr. Green stated that the heart showed hypertrophy of the fibers in the left ventricle and he saw interstitial myocardial fibrosis in the subendocardial region and that those hypertrophic changes in themselves would not be related to coal mine dust exposure. EX-3 at 99. Dr. Green reiterated his prior testimony that there was no evidence of left ventricular failure because the miner did not have any evidence of persistent left ventricular failure in the form of heart failure cells, or pulmonary edema, which is a sign of acute left ventricular failure. Dr. Green stated that the miner's nocturnal breathing problems could be due to right heart disease, a problem in the lungs, or left heart failure. EX-3 at 106.

Dr. Green testified that he and Dr. Perper differed in their opinions as to the severity of sclerosis of the small interpulmonary vessels, although Dr. Perper did not identify the plexiform lesions, which were his basis for diagnosing the more advanced forms of pulmonary hypertension. EX-3 at 108–109. Dr. Green stated that diabetes could worsen the miner's vascular disease, that left ventricular changes would not be associated with pneumoconiosis, and that left ventricular hypertrophy can cause a fatal heart arrhythmia. EX-3 at 109. Dr. Green acknowledged that no one is ever going to know for sure where the electrical trigger started that killed the miner and that he could not state where that trigger was within a reasonable degree of medical certainty, but that his opinion is that given the miner's severe right-sided disease, that is the most likely cause; even if the event originated in the left side of the heart, the right-heart disease might well have contributed to it. EX-3 at 111. Dr. Green testified that he can state with a reasonable degree of medical certainty that it was the right heart that caused the cardiac arrest. EX-3 at 125.

Dr. Joshua A. Perper[7]

Dr. Perper reviewed the records on behalf of the Department of Labor and wrote a report dated June 20, 2002, which appears in the record at DX-54. Dr. Perper is a forensic pathologist and attorney and reviewed the miner's autopsy slides, medical reports, death certificate and the medical reports of Drs. Elnicki, Green, Kleinerman, Tuteur, and Oesterling. Dr. Perper's microscopic diagnoses are: 1) coal workers' pneumoconiosis, macular, minimal; 2) centrilobular emphysema, moderate; 3) sclerosis of small intra-pulmonary vessels, slight to moderate; 4) congestion and edema of the lungs; 5) focal myocardial fibrosis and hypertrophy of myocardial fibers, consistent with hypertrophic cardiomyopathy; 6) benign hyperplasia of prostate; 7) congestion of internal organs.

Dr. Perper opined that the miner had evidence of minimal, simple pneumoconiosis, macular, with no evidence at all of pneumoconiotic micronodules or macronodules. He further opined that because of the scarcity of pneumoconiotic macules, the diagnosis of coal workers' pneumoconiosis is "rather liberal." Dr. Perper stated that the miner's coal workers' pneumoconiosis was a result of occupational exposure to coal mine employment, as substantiated by the anthracotic pigment and small numbers of birefringent silica crystals. Dr. Perper opined that the miner's simple coal workers' pneumoconiosis was only minimal and

[7] Dr. Perper's credentials are not in the record; however, I take judicial notice that he is a forensic pathologist and attorney.

macular, too mild to contribute to his death or to be a hastening factor in his death. He explained that although it is true that coal workers' pneumoconiosis can result in centrilobular emphysema, it is unreasonable to assume that the miner's minimal, almost non-existent CWP could result in any degree of centrilobular emphysema. Dr. Perper opined that the miner's centrilobular emphysema was related to his cigarette smoking history. In addition, Dr. Perper noted that the miner did not die with preceding respiratory symptomatology but following acute cardiac dysrhythmia with ventricular fibrillation.

Dr. Perper stated that he agrees with Dr. Green that the miner had very mild coronary disease and a questionable history of hypertension that cannot explain the fatal arrhythmic attack. He noted that the miner had a very enlarged heart and that the microscopic exam showed both hypertrophic myocardial changes and significant interstitial focal myocardial fibrosis—indicating a diagnosis of hypertrophic cardiomyopathy—which is often idiopathic and occurs in the absence of hypertensive cardiovascular disease. Dr. Perper stated that such cardiomyopathy results in fatal abnormal conduction of myocardial electrical impulses.

Dr. Perper concluded within a reasonable degree of medical certainty that: 1) the miner had evidence of minimal and scarce, macular, simple coal workers' pneumoconiosis; 2) the coal workers' pneumoconiosis was a result of occupational exposure to mixed coal dust; 3) the coal workers' pneumoconiosis was too mild to be a substantial or significant cause of death, a contributory cause of death or a hastening factor in the miner's death; 4) the death was due to a cardiac dysrhythmia (documented ventricular fibrillation) on the backround of hypertrophic cardiomyopathy with cardiomegaly and focal myocardial fibrosis.

Dr. D. Michael Elnicki

Dr. Elnicki, board certified in internal medicine, submitted a second report dated September 12, 2002, which appears in the record at CX-1. His curriculum vitae appears in the record at CX-2. Dr. Elnicki stated that he reviewed the letter he wrote Mr. Bonfante in 1997, the autopsy report, the miner's medical records from the West Virginia University Medical Center, and the reports of Drs. Perper, Tuteur, Oesterling, Kleinerman, and Green. Dr. Elnicki stated that he was the miner's primary care physician and that he treated the miner primarily for type II diabetes mellitus, which was controlled by lifestyle measures alone. He generally saw the miner every three to four months. He noted that the miner kept his blood pressure within normal range and was able to discontinue diuretic therapy. Dr. Elnicki noted that he saw the miner for pulmonary issues, beginning in 1994 with a follow-up x-ray as part of a black lung evaluation. He noted that the repeat film did not show any significant lung pathology. Dr. Elnicki stated that the miner occasionally mentioned dyspnea, associated with strenuous activity or physiologic stress, such as his Rocky Mountain trip. He noted that the miner did not complain of chest pain, palpitations, or other cardiovascular complaints.

Dr. Elnicki stated that the bulk of the testing the miner had at his clinic consisted of hemoglobin for diabetes, audiology, two chest x-rays—the first for black lung and the second as a preoperative measure prior to knee surgery. He had a CT scan of the head following his TIA. Other laboratory data included thyroid function tests, blood counts, kidney function, urinalysis, clotting studies, and liver function tests, all of which were normal. Dr. Enicki noted that the

miner had a pre-op electrocardiogram that showed a sinus brachycardia and non-diagnostic Q-waves in an inferior pattern, but that there was not evidence of either left or right ventricular hypertrophy. Dr. Elnicki noted that at his last visit, the miner's lung and heart exams were essentially normal, which he stated was often the case, even in advanced emphysema.

In relation to the cause of the miner's cardiac arrest, Dr. Elnicki first discussed what did not cause it. He noted that based on autopsy: 1) the miner did not have a myocardial infarction and none of the plaque lesions present were hemodynamically significant; 2) there was no evidence of a cerebral vascular event (stroke); 3) he did not have a pulmonary embolism; 4) he did not have a peripheral vascular catastrophe such as a ruptured aortic aneurysm; 5) there was no evidence of any infection or malignancy. Dr. Elnicki then noted abnormalities found on pathologic exam. He stated that on gross examination of the lungs the miner had emphysematous blebs, nodules, bullae and black anthracotic material. Examination of the heart was consistent with mild left ventricular hypertrophy and moderate to severe right ventricular hypertrophy in the heart. He stated that the lungs showed gross evidence of emphysema and that microscopic evaluation revealed simple coal workers' pneumoconiosis.

Dr. Elnicki noted that the miner died a sudden death, most likely of an acute cardiac arrhythmia. He explained that although the left ventricular hypertrophy is mild, it is associated with ventricular arrhythmias, which can cause sudden death. In addition, he explained that right ventricular hypertrophy is also associated with supraventricular and ventricular arrhythmias. Dr. Elnicki cited a passage of the textbook *The Heart*, which notes that advanced right ventricular hypertrophy is often present without ECG findings. In addition, the same text also describes nonspecific symptoms associated with advanced pulmonary hypertension including transient cerebral ischemia, syncope, and dyspnea particularly with exposure to altitude. Dr. Elnicki opined that the miner's advanced right ventricular hypertrophy is likely the cause of his unexplained TIA and his difficulty on his Rocky Mountain trip. Dr. Elnicki stated that the miner's history of well-controlled hypertension would be the most likely explanation of his left ventricular hypertrophy, and his right ventricular hypertrophy would most likely be due to his emphysema, rather than his left ventricular hypertrophy. Dr. Elnicki opined that the miner's emphysema was more a result of his nearly 40 years of underground coal work, as opposed to his relatively light smoking history (12 pack years).

Dr. Elnicki stated that the miner died of sudden cardiac death most likely due to an arrhythmia. He stated that it is impossible to say whether the arrhythmia is directly due to a left versus right ventricular hypertrophy, since both are associated with arrhythmias, but his advanced state of pulmonary pathology makes that a leading suspect for his symptoms prior to his death. Dr. Elnicki stated that the weight of the miner's occupational exposure is far greater than that of his remote smoking history. Dr. Elnicki opined that the miner's pulmonary disease played a significant role in his death and the cause of his pulmonary disease was largely his occupational exposure to materials he encountered as a coal miner.

Dr. Everett F. Oesterling

The medical report of Dr. Oesterling is dated November 8, 2002 and appears at EX-1. Dr. Oesterling is board certified in anatomical and clinical pathology and his report is based on a

review of the reports of Drs. Green and Perper. Dr. Oesterling, responding to Dr. Green's comment that his committee report did not specify a minimum size for a coal dust macule, noted that the article stated "…these early lesions will show up as black macules of 1 to 4 mm in diameter." Dr. Oesterling noted that developing lungs, which would include the miner's smoking period, are far more susceptible to the damages of cigarette smoke and that once emphysema is present, the alveolar membranes have ruptured and do not regenerate. Emphysema, he explained, is thus a lifelong condition. Dr. Oesterling stated that he agrees with Dr. Perper's assessment that "it is unreasonable to assume that the minimal, almost nonexistent coalworkers' pneumoconiosis (in the miner) could result in any degree of centrilobular emphysema." Dr. Oesterling also agreed with Dr. Perper that the miner's coalworkers' pneumoconiosis was too mild to be a substantial, significant, contributory, or hastening cause of death. Dr. Oesterling restated that the miner had no disease arising out of mine dust exposure, for the very mild dust deposition in his lungs was insufficient to have altered pulmonary function, thus it did not in any way hasten, contribute to, or cause his death.

Dr. Oesterling's deposition from the prior litigation appears at EX-6. This deposition was taken on October 27, 1998. Dr. Oesterling testified that he is board certified in anatomical and clinical pathology and nuclear medicine. EX-6 at 3. Dr. Oesterling testified that the lung slides he received were representative of the lung as a whole and that the quality of the slides he gets from Dr. Wecht are always excellent. EX-6 at 8–9. Dr. Oesterling testified that to make the diagnosis of coal workers' pneumoconiosis, the lesions should exceed a millimeter in size, be accompanied by areas of focal emphysema, and contain silica crystals within the dust. EX-6 at 11. Dr. Oesterling testified that there were not sufficient changes in the functioning part of the lung to warrant a pathologic diagnosis of coal workers' pneumoconiosis; the largest parenchymal lesion he saw was less than a millimeter and he did not consider this to be of adequate size to warrant a diagnosis of pneumoconiosis. EX-6 at 12, 39. Dr. Oesterling testified that the areas of minimal focal emphysema he saw were not enough to alter lung function and he does not think the focal emphysema was significant in this case. EX-6 at 14. Dr. Oesterling stated that he thinks the miner's smoking history at a relatively young age accounted for some of the emphysema, in combination with the aging process which includes progressive emphysema. EX-6 at 20. Dr. Oesterling stated that the centrilobular emphysema was not the result of coal mine dust exposure; he said that studies in this country have shown that centrilobular emphysema is of no greater incidence in miners than in the general population, excluding other factors. EX-6 at 21.

Dr. Oesterling stated that the miner had fairly marked passive congestion of the lungs at the time of death, indicating a failing left ventricle. EX-6 at 25. Dr. Oesterling stated that photographs of the spleen indicate that the miner was experiencing right-sided failure as well. EX-6 at 27. Dr. Oesterling stated that the miner was not suffering from cor pulmonale, in that both ventricles were hypertrophied which is commonly seen in patients experiencing ischemic, left ventricular disease and the right side of the heart has to work harder to compensate so that both sides will enlarge. EX-3 at 28. Dr. Oesterling stated that coal dust exposure did not play a role in the right ventricle enlargement because he did not see changes within the small pulmonary arteries that reflected prolonged pulmonary hypertension which he would expect to see with true cor pulmonale. *Id*. Dr. Oesterling stated that the miner had fairly diffuse ischemic heart disease involving his left ventricle and that the changes in the miner's lungs were

insufficient to have altered oxygenation enough to attribute this to coal dust exposure. EX-6 at 30.

Dr. Oesterling opined that the miner died of cardiac arrest due to atrial fibrillation that was uncontrolled and biventricular failure. He opined that neither coal worker's pneumoconiosis nor the miner's exposure to coal dust substantially contributed to or hastened his death. EX-6 at 33. Dr. Oesterling acknowledged that a person can have cor pulmonale and hypertrophy of both ventricles due to fibrosis in the lung but said that the miner did not have fibrosis within the lung. EX-6 at 44. Dr. Oesterling stated that he would not expect to see emphysema in coal miners unless there is encroachment on the airways due to the fibrotic process of nodules or progressive massive fibrosis, which the miner did not have. EX-6 at 52.

Dr. Richard L. Naeye

Dr. Naeye reviewed the autopsy slides, Claimant's medical records, and the reports of Drs. Wecht, Elnicki, Oesterling, Tuteur, Green, Perper, and Kleinerman. Dr. Naeye's report appears at EX-2. Dr. Naeye's curriculum vitae is not in the record; however, at his deposition he testified that he is board certified in anatomic and clinical pathology. EX-4 at 5. On examination of the slides, Dr. Naeye noted that there is a small to moderate amount of black pigment at several subpleural sites. There are a few birefringent crystals of non-toxic silicates admixed with the pigment. He noted that at several loci there is a small amount of fibrous tissue associated with the pigment, but it is far from certain that this fibrosis has an occupational origin because there are other subpleural sites where such fibrosis is present without accompanying black pigment. Dr. Naeye noted that otherwise there is only a very small amount of black pigment in the lung tissues, mainly adjacent to small arteries and airways and only rarely is there any fibrosis associated with pigment. Dr. Naeye explained that, when such fibrosis is present in the miner's lungs, it is not accompanied by any very tiny, birefringent crystals of toxic-free silica, which raises doubt about whether the fibrosis in the two previously mentioned anthracotic macules had an occupational origin. Dr. Naeye stated that in such cases he gives the benefit of the doubt to the miner and diagnoses very mild, simple CWP. Dr. Naeye stated that taken together, the two macules in the lung occupy so little lung tissue that there is no way they could have affected lung function, caused disability or hastened death. Dr. Naeye explained that their lack of clinical importance is reinforced by the fact that a regional lymph node has only a small amount of black pigment with no associated fibrosis and the lack of fibrosis is important because the major fibrogenic agent in coal mine dust is free silica. Such silica usually remains far longer in lymph nodes than in lung tissues and as a result, in individuals who have CWP, fibrosis is usually more advanced in regional lymph nodes than in nearby lung tissues. Dr. Naeye opined that overall, centrilobular emphysema is mild in the miner, far milder than is present in the lungs of most bituminous coal miners. He noted that there is no microscopic evidence of chronic bronchitis and that microscopic review of heart tissues disclosed the presence of small fibrotic foci in one piece of tissue from the wall of the left ventricle which is a possible site of origin of the fatal cardiac arrhythmia.

Dr. Naeye concluded that the miner had the minimal findings required to diagnose coal workers' pneumoconiosis but that the lesions are far too few in number and small in size to have had any measurable influence on lung function. In addition, pulmonary function studies

conducted in 1994 produced normal FEV1 and FVC values, the normal ratio of mucous to serous glands in the lungs are very strong evidence that the miner did not experience chronic bronchitis at the time of death, shortly before death the miner reported dyspnea only with strenuous activity, and the centrilobular emphysema is too mild to have caused any measurable abnormalities in lung function. Dr. Naeye opined that in toto, the evidence is very strong that the miner had no clinically significant disorders in his lungs and the focal areas of fibrosis in the left ventricular wall of the heart are a plausible site from which his fatal cardiac arrhythmia could have arisen.

Dr. Naeye was deposed on March 26, 2003.[8] Dr. Naeye testified that he is on the staff of the Milton S. Hershey Medical Center for Penn State University College of Medicine. EX-4 at 5. Dr. Naeye stated that he was appointed to the original committee of the American College of Pathologists to identify the components of coal workers' pneumoconiosis and in conjunction with this work the committee published a paper 30 years ago that has never been superseded by any significant changes. EX-4 at 6. Dr. Naeye testified that he was active in researching lung diseases of coal miners in the early 1970s and that his research since that time has centered on fetal and infant development. Dr. Naeye testified that regularly reads JAMA and the New England Journal of Medicine. EX-4 at 8.

Dr. Naeye testified that he reviewed the miner's medical records, autopsy slides, and Dr. Green's deposition testimony, and noted that the miner worked underground for 40 years, injured his shoulder and stopped mining. He stated that the miner smoked one to two packs of cigarettes per day for 21 years, which would make a 31-pack-year history. Dr. Naeye testified that he does not recall where in the medical records he saw that figure. EX-4 at 10–11, 47–48. Dr. Naeye noted that the miner had type II diabetes, sometimes controlled by diet, and had a sudden death in 1997 at age 57. EX-4 at 11. Dr. Naeye testified that the miner's 1994 pulmonary function study values were very good for a man of his age with a 21-pack-year smoking history. In addition, Dr. Naeye stated that the miner's arterial blood gas study was normal and that the miner did not report any pulmonary symptomatology in connection with his fatal event. EX-4 at 12.

Dr. Naeye testified that he received 29 slides—ten with lung tissue and at least fifteen with heart tissue—and that the slides contained enough pathologic evidence to identify the disease processes present. EX-4 at 12–14) Pertaining to the lung tissue slides, Dr. Naeye testified that there was a small to moderate amount of black pigment in the lung at subpleural sites and that at several of those sites there were a few birefringent crystals mixed with the pigment. EX-4 at 14. He explained that these birefringent crystals were nontoxic because they were too large to be toxic free silica. *Id*. Dr. Naeye stated that with the black pigment was a small amount of fibrous tissue and that he was not sure it was all related to occupational exposure to mine dust, but that his policy is to give the benefit of the doubt to the miner. Therefore, he stated that the small amount of fibrous tissue did have some occupational origin.

[8] At the hearing and deposition, Claimant's attorney objected to two areas of Dr. Naeye's testimony as being outside the scope of his report. Specifically, Claimant objects to Dr. Naeye's opinion of cor pulmonale and his discussion of abnormalities in the microcirculation of the heart. Tr. 12–19; EX-4 at 31. After reviewing the testimony and the record, I find that Dr. Naeye's statements pertaining to cor pulmonale are admissible because he reviewed Dr. Green's testimony, which is centered on cor pulmonale, in preparation for this deposition. However, I will not allow the testimony pertaining to the microcirculation of the heart because it is well outside the scope of his report and not a factor in preparing for this deposition.

EX-4 at 14–15. Dr. Naeye testfied that he diagnosed coal workers' pneumoconiosis and does so whenever he finds fibrosis with black pigment, even in very small amounts. EX-4 at 15–16. Dr. Naeye stated that the coal workers' pneumoconiosis was very mild and the level of disease was macular. EX-4 at 16.

Dr. Naeye explained that there is always a small degree of uncertainty about the nature of fibrous tissue associated with black pigment in the subpleural areas because, when small amounts of pneumonia or pleurisy heals, sometimes fibrous tissue is found beneath the pleura. EX-4 at 17. Dr. Naeye testified that he agreed with Dr. Perper's assessment that because of the scarcity of pneumoconiotic macules, the CWP diagnosis is liberal. EX-4 at 18. Dr. Naeye testified that, based on his review of the miner's lifetime records, the coal workers' pneumoconiosis was not of clinical significance. Further, the physician testified that this was confirmed by the pulmonary function and blood gas studies. EX-4 at 19. Dr. Naeye stated that although Dr. Wecht identified coal dust nodules measuring three to four millimeters, he himself did not. *Id*.

Dr. Naeye testified that centrilobular emphysema was also present, but that coal mine dust only has a very small role in that process as opposed to cigarette smoke, which is the dominant cause; the physician reiterated that the miner had a 31-pack year history of smoking. EX-4 at 20. He stated that that does not exclude coal dust exposure from having a few percent role in the presence of the centrilobular emphysema. *Id*. Dr. Naeye stated that the miner did not have chronic bronchitis, which is unusual for coal miners. EX-4 at 20–21. Dr. Naeye testified that there is a lot of individual variation in susceptibility to developing chronic bronchitis and centrilobular emphysema. EX-4 at 21. Dr. Naeye stated that Dr. Wecht's description of moderate to severe emphysema would have no effect on his own opinions because the pulmonary function studies did not reveal it was severe and because pathologists take tissues from the most diseased parts of the lungs they can find, and the emphysema looks worse on slides than reflected in pulmonary function studies. EX-4 at 22–23. Dr. Naeye testified that he was convinced coal mine dust exposure can cause centrilobular emphysema but he finally concluded that it can have a small role, not a large role. EX-4 at 24. Dr. Naeye stated that the miner's coal mine dust exposure was so mild that it did not have any clinical significance and that the miner's coal mine dust exposure was not a contributing factor to his developing centrilobular emphysema. *Id*. He stated that the miner's cigarette smoking primarily led to the centrilobular emphysema because it is the commonest cause in all populations in the world and in the United States. EX-4 at 24–25.

Dr. Naeye stated that, unlike Dr. Green, he does not think it is true that when a person stops smoking before age 40, he almost never develops centrilobular emphysema from smoking because there are enormous genetic factors as well as occupational exposures that govern whether somebody develops it. EX-4 at 25. Dr. Naeye stated that he was not sure whether Dr. Green provided a citation to this opinion and whether he would check the citation would depend on whether he had enough time to do so. EX-4 at 48–49.

Dr. Naeye stated that the miner's centrilobular emphysema was not of clinical significance based on his description of the miner's later life and it is uncommon to have clinically significant emphysema without chronic bronchitis. EX-4 at 25–26. Dr. Naeye stated

that he had never before heard Dr. Green's opinion that coal workers' pneumoconiosis macules break down and become harder to recognize in the presence of emphysema, and that they do not break down when there is fibrous tissue. EX-4 at 26–27. Dr. Naeye stated that the miner's emphysema did not have a role in his death as demonstrated by the fact that the miner's pulmonary function studies were normal and he did not have chronic bronchitis. EX-4 at 27.

Dr. Naeye testified that the heart slides revealed a small area of fibrosis in the myocardium, with the major abnormality being great thickening of the walls of arterioles and small arteries. EX-4 at 27. This happens to cigarette smokers, and there is an excess of sudden death in cigarette smokers due to destruction of normal muscle tissue. EX-4 at 27–28. Dr. Naeye testified that the miner's smoking history does not correlate with his sudden death because he had stopped smoking several years before, but the damage had apparently already occurred. EX-4 at 28–29.

Dr. Naeye testified that he disagrees with Dr. Wecht's diagnosis of cor pulmonale because he found from past experience that Dr. Wecht did not measure the thickness of the ventricles from the base of the trabeculae. EX-4 at 33. He further stated that he is not sure what Dr. Wecht's procedures are now and as he did not see the heart himself, he cannot testify beyond that. *Id*. Dr. Naeye stated that his opinion, within a reasonable degree of medical certainty, is that the miner had a sudden cardiac arrhythmia which caused death as a result of sclerotic lesions within the myocardium of the heart. *Id*. Dr. Naeye stated that neither coal mine dust exposure nor pneumoconiosis substantially contributed to the miner's death nor hastened his death. EX-4 at 34–35.

On cross-examination, Dr. Naeye reiterated that he found two anthracotic macules of less than one millimeter and that some doctors would not diagnose pneumoconiosis because the lesions were less than one millimeter. EX-4 at 44. Dr. Naeye testified that he diagnosed mild centrilobular emphysema based on the slides and pulmonary function studies, and that the miner's pneumoconiosis had no measurable effect because the lesions were small and he did not suffer from chronic bronchitis. EX-4 at 45. Dr. Naeye stated that he could not say which report indicated the miner smoked for 21 years as opposed to thirteen. EX-4 at 47–48.

Dr. Naeye reiterated that coal dust had only a small percentage role in emphysema as compared to cigarette smoke, about five percent, and he is not sure from where he got that figure. EX-4 at 63, 65. When asked whether the number of years a miner was exposed to coal dust contributed to centrilobular emphysema, Dr. Naeye stated that he did not think there is any information available that would make it possible to make a definitive statement. EX-4 at 65. Dr. Naeye acknowledged that had no idea that Dr. Green was researching that issue or that he had published articles in this area. *Id*. He testified that his opinion is that there are enormous differences in the composition of coal dust from mine to mine and genetic differences in susceptibility that it is impossible to quantify. *Id*. When asked, Dr. Naeye agreed that in 1972, in his early work related to a JAMA article, he concluded that centrilobular, panlobular, and another form of emphysema were part of the black lung process. EX-4 at 68–70. He explained that at that time, the composition of coal mine dust was different and that centrilobular emphysema was part of pneumoconiosis in hard-coal miners but not soft-coal miners. EX-4 at 70.

Dr. Naeye testified that it would be possible for a miner to develop clinically significant emphysema without progressive massive fibrosis, but 92 percent of miners smoke cigarettes and almost all of them have emphysema. EX-4 at 74. He further testified that he would attribute all but about five percent of that emphysema to cigarette smoking, with genetic susceptibility being another major factor. *Id*. Dr. Naeye stated that the miner's heart disease was not related to his coal mine dust exposure and that the miner's centrilobular emphysema was mild and was no factor whatsoever in causing his death. EX-4 at 75.

Dr. Jerrold L. Abraham

Dr. Abraham, board certified in anatomic pathology wrote a report dated May 30, 2003 that appears in the record at CX-7. Dr. Abraham reviewed the miner's medical records and pathology slides. Dr. Abraham stated that the sections of lung tissue show definite pneumoconiosis. He further stated that there is accumulation of dust particles of a mixed nature, associated with macular, interstitial, and subpleural fibrosis. Dr. Abraham explained that because there is fibrosis associated with the macules, they may be termed early fibrotic lesions. Dr. Abraham noted that the dust in the macrophages in the lung consists of a mixture of opaque particles—some coarse and some very fine—and a large number of birefringent particles. He noted that some of the birefringent particles are typical of crystalline silica, and others have the strong birefringence typical of aluminum silicates, which are all consistent with dust originating from coal mining, including crystalline silica. Dr. Abraham also commented that there were numerous iron-rich particles present, which are consistent with exposure to welding or similar metal-working operations. He noted that he did not observe any asbestos bodies in the H&E stained sections. Dr. Abraham stated that his other major observations are the presence of focally severe pulmonary arteriolar thickening, consistent with significant pulmonary arterial hypertension, and mild centrilobular emphysema. He explained that this correlates with the finding of the miner's right ventricular thickening, and supports the diagnosis of cor pulmonale.

On review of the medical records, Dr. Abraham noted that the exact cause of the miner's fatal cardiac arrhythmia was not clear, but that cor pulmonale is a factor which predisposes one to cardiac arrhythmia. He further explained that factors responsible for cor pulmonale are lung and cardiac disease. Dr. Abraham noted that the miner had definite and clear evidence of pneumoconiosis but that the relationship of the severity of the pneumoconiosis seen in a few microscopic sections to the disease symptomatology is not simple to quantify. Dr. Abraham stated that he can confirm the miner had coal workers' pneumoconiosis, with the extent reflected in the available slides being mild. He also stated that the cor pulmonale is "quite significant."

Dr. Abraham concluded, with a reasonable degree of medical certainty, that the miner's coal mine employment was the source of the dust exposure in his lungs at autopsy, the cause of his pneumoconiosis, and was a substantial contributing cause of his cor pulmonale and death. Dr. Abraham further explained that the miner's coal mine employment was the source of the dust he inhaled and the source of his pneumoconiosis. Additionally, the physician explained that the coal workers' pneumoconiosis contributed to the miner's cor pulmonale because any lung disease involving the loss of functional lung tissue makes it difficult to get oxygen into the bloodstream, which increases pulmonary arterial blood pressure as the heart tries to send more blood into the lungs. Dr. Abraham stated that the miner's coal dust exposure contributed to his

emphysema which also causes pulmonary arterial hypertension and that crystalline silica has been shown to promote the development of emphysema. Finally, Dr. Abraham explained that cor pulmonale contributed to the miner's fatal cardiac arrhythmia because cor pulmonale indicates a failure of the heart to keep up with the demands of the lung which leads to the following cycle:

> [a] need for more oxygen → need for more blood to the lungs → increased thickness of right ventricle of heart → increased pulmonary artery pressure→ increased thickness of pulmonary artery walls → increased resistance to pulmonary artery blood flow → need for more pressure → etc., terminating in insufficient blood (oxygen) getting to the heart muscle and a terminal cardiac event such as an arrhythmia.

Dr. Abraham was deposed on September 11, 2003. EX-7. Dr. Abraham testified that he is a pathologist with an interest in the pathology related to occupational lung disease. He stated that in addition to being a professor of pathology at the State University of New York in Syracuse and the Director of Environmental and Occupational Pathology at the University of Syracuse, he worked for the U.S. Public Health Service at NIOSH and administered the National Coal Workers' Autopsy Study. Dr. Abraham testified that he determines questions related to cause of death nearly every day. Dr. Abraham enumerated, in his deposition testimony, the specific medical records and slides that he reviewed in connection with this claim.

Dr. Abraham's testimony was consistent with his report as he explained the basis for his diagnosis of cor pulmonale. He explained that, based on the pathology, the miner's pulmonary arterial hypertension was a result of pneumoconiosis and emphysema and the cause of the emphysema was a mixture of the miner's smoking and coal mine dust exposure. EX-7 at 21. Dr. Abraham further testified that he cannot determine what percent of the emphysema was caused by smoking, as opposed to coal mine dust exposure, but it showed features of both factors. EX-7 at 23, 39. Dr. Abraham explained that cor pulmonale means heart disease caused by lung disease, which relates to pulmonary hypertension. In addition, he explained that damage to the lung makes the right side of the heart work harder, which causes the muscles of the heart to enlarge, leading to right ventricular hypertrophy. EX-7 at 23–24. Dr. Abraham testified that the miner's death was caused by a cardiac arrhythmia due to lung disease, which reduced the ability of oxygen to reach his bloodstream and heart. EX-7 at 25, 29.

On cross-examination, Dr. Abraham testified that he did not see any nodules, did not record the number, size, or extent of the macules he saw or whether the macular changes in and of themselves or the emphysema would be sufficient to alter pulmonary function. EX-7 at 36–37. In addition, Dr. Abraham acknowledged that his report did not discuss or reference the clinical objective data gathered during the miner's lifetime, such as arterial blood gas studies, and whether these studies would support his conclusions. EX-7 at 37. In addition, Dr. Abraham stated that he did not discuss the miner's pulmonary function abilities during his lifetime and did not review his records or tests to determine whether the miner had hypoxemia during his lifetime. EX-37 at 45–49. Dr. Abraham testified that the extent of both the pneumoconiosis and the emphysema were mild. EX-7 at 39. Dr. Abraham clarified that cardiac disease does not cause cor pulmonale—as the statement in his report is unclear—and reiterated that cor

pulmonale is cardiac disease caused by lung disease. EX-7 at 41. Dr. Abraham further acknowledged that he did not mention any abnormalities in the heart muscle itself; specifically, fibrosis in the myocardium, blockage of the coronary arteries, left-sided cardiac enlargement, failure, or ischemia, edema of the lungs, or passive congestion of the lungs, arteriosclerotic or hypertensive heart disease, biventricular hypertrophy, or food aspiration. EX-7 at 41–43, 47. Dr. Abraham stated that the exact cause of the cardiac arrhythmia was not clear, without being there at the time it occurred. EX-7 at 43. Dr. Abraham stated that he did consider left-sided heart disease, but did not mention it in his report because he concluded that the coal mining was a substantial contributing cause of the miner's cor pulmonale and death—as opposed to the only cause. EX-7 at 50–51.[9]

CONCLUSIONS OF LAW

Length of Coal Mine Employment

The parties stipulated and I find that the miner was a coal miner within the meaning of the Act for at least 34.4 years. Tr. 33.

Date of Filing

Claimant filed her claim for modification October 31, 2001 within one year from the prior denial. Tr. 33.

Responsible Operator

The parties have stipulated and I find that Shannopin Mining Company is the responsible operator and will provide payment of any benefits awarded to Claimant. Tr. 33.

Eligible Survivor

Claimant has not remarried and is eligible for benefits under the Act as she is the surviving spouse of the miner. Tr. 32–33.

Pneumoconiosis

Employer has stipulated to the existence of pneumoconiosis arising from coal mine employment; thus, I find that the miner suffered from pneumoconiosis. Tr. 33.

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[9] Employer objected to this testimony as being beyond the scope of the medical report, direct, and cross-examination. After re-reading the pertinent testimony, I find that Dr. Abraham's statements are within the scope of Employer's cross-examination questions. Employer also objected to Dr. Abraham's testimony relating to the miner's blood gas studies to a progression to cor pulmonale and requested that this testimony be stricken from the record. EX-7 at 54–55. I find that this testimony does exceed the scope of the medical report, direct, and cross-examinations. Employer's objection is granted, in part, and this portion of testimony will be stricken from the record.

Modification

Any party to a proceeding may request modification at any time before one year from the date of the last payment of benefits or at any time before one year after the denial of a claim. 20 C.F.R. § 725.310(a). Upon the showing of a "change in conditions" or a "mistake in a determination of fact" the terms of an award or the decision to deny benefits may be reconsidered. 20 C.F.R. § 725.310. As there can be no change in the deceased miner's condition, Claimant must demonstrate that a mistake of fact was made in the prior determination of this claim.

In *O'Keeffe v. Aerojet-General Shipyards, Inc*., 404 U.S. 254, 257 (1971), the U.S. Supreme Court held that an administrative law judge should review all evidence of record to determine if there has been, with respect to a request for modification, a mistake in a determination of fact.[10] In considering a motion for modification, the administrative law judge is vested "with broad discretion to correct mistakes of fact, whether demonstrated by *wholly new evidence*, cumulative evidence, or merely further reflection on the evidence initially submitted" (emphasis added). *See also Jessee v. Director, OWCP,* 5 F.3d 723 (4th Cir. 1993); *Director, OWCP v. Drummond Coal Co*. (*Cornelius*), 831 F.2d 240 (11th Cir. 1987). Additionally, in *Keating v. Director, OWCP*, 71 F.3d 1118 (3rd Cir. 1995), the Third Circuit held that, on modification, "the [ALJ] must review all evidence of record—any new evidence submitted in support of modification as well as the evidence previously of record—and 'further reflect' on whether any mistakes [of] fact were made in the previous adjudication of the case."

Judge Leland's initial decision awarding benefits was remanded after the Benefits Review Board held that he improperly accorded greater weight to Drs. Wecht and Elnicki, solely on the basis of their positions as the autopsy prosector and treating physician, respectively. On remand, Judge Leland found that the opinions of Drs. Oesterling and Kleinerman were better reasoned than Dr. Wecht's and that Dr. Kleinerman's credentials were better than Dr. Wecht's. In addition, Judge Leland held that Dr. Tuteur's opinion was less equivocal and better reasoned than Dr. Elnicki's, and that Dr. Elnicki's credentials were not in the record. After reviewing and reconsidering all of the evidence in the record before Judge Leland, in addition to the new evidence submitted, I find that that there a was a mistake in the "ultimate fact of entitlement." *Keating v. Director, OWCP*, 71 F.3d at 1123. Specifically, I find that the previous decision should be modified because the more credible evidence now shows that pneumoconiosis contributed to the miner's death.

Death Due to Pneumoconiosis

Claimant's application for benefits was filed on June 30, 1997 and is governed by the Part 718 Regulations. Benefits are provided to the eligible survivors of a miner who can establish the miner died as a result of pneumoconiosis. 20 C.F.R. § 718.205(a). Death will be considered due to pneumoconiosis if any of the following criteria is met:

[10] Although this case interpreted language from the Longshoremen's and Harbor Workers' Compensation Act, 33 U.S.C. 922, the Longshore regulations are incorporated into the Black Lung Benefits Act by 33 U.S.C. 932(a) and provide statutory authority to modify orders and awards.

(1) Where competent medical evidence establishes that the miner's death was due to pneumoconiosis, or
(2) where pneumoconiosis was a substantial contributing cause or factor leading to the miner's death or where death was caused by complications of pneumoconiosis, or
(3) where the presumption in § 718.304 is applicable.[11]

20 C.F.R. § 718.205(c).

The Regulations further provide that survivors are not eligible for benefits if the miner's death was caused by a traumatic injury or if the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death. 20 C.F.R. § 718.205(c)(4). The Third Circuit, when this case arose, held that if pneumoconiosis hastens the miner's death, it is a substantially contributing cause of death for the purposes of 20 C.F.R. § 718.205(c)(4). *Lukosevicz v. Director, OWCP*, 888 F.2d 1001 (3rd Cir. 1989).

The death certificate lists the causes of death as ventricular fibrillation and arteriosclerotic heart disease. Chronic obstructive pulmonary disease was listed as another significant condition contributing to death. A death certificate, in and of itself, is an unreliable report of the miner's condition. *Smith v. Camco Mining, Inc.*, 13 B.L.R. 1017 (1989); *Addison v. OWCP*, 11 B.L.R. 1-68 (1988).

It is not disputed that the miner suffered from simple coal workers' pneumoconiosis at the time of his death. Nine physicians rendered opinions as to whether and to what extent pneumoconiosis contributed to or hastened the miner's death pursuant to § 725.205(c). Drs. Wecht, Green, Abraham, and Elnicki concluded that pneumoconiosis contributed to or hastened the miner's death. Drs. Oesterling, Kleinerman, Tuteur, Perper, and Naeye determined that pneumoconiosis did not contribute to, cause, or hasten the miner's death. The medical evidence in this case is extensive and complex. The crux of the matter is centered on whether the miner suffered from cor pulmonale, caused by his pneumoconiosis and emphysema, which then resulted in a fatal cardiac arrhythmia and death.

Standard of Review

The administrative law judge need not accept the opinion of any particular medical witness or expert, but must weigh all the evidence and draw his/her own conclusions and inferences. *Lafferty v. Cannelton Industries, Inc.*, 12 B.L.R. 1-190 (1989); *Stark v. Director, OWCP*, 9 B.L.R. 1-36 (1986); *Todd Shipyards Corp. v. Donovan*, 300 F.2d 741 (5th Cir. 1962). The adjudicator's function is to resolve the conflicts in the medical evidence; those findings will not be disturbed on appeal if supported by substantial evidence. *Lafferty,* 12 B.L.R. 1-190; *Fagg v. Amax Coal Co.*, 12 B.L.R. 1-77 (1988), *aff'd*, 865 F.2d 916 (7th Cir. 1989); *Short v. Westmoreland Coal Co.*, 10 B.L.R. 1-127 (1987); *Piccin v. Director, OWCP*, 6 B.L.R. 1-616 (1983); *Peabody Coal Co. v. Lowis*, 708 F.2d 266, 5 B.L.R. 2-84 (7th Cir. 1983).

[11] The presumption at § 718.304 is not applicable in the present case.

In considering the medical evidence of record, an administrative law judge must not selectively analyze the evidence. *See Wright v. Director, OWCP*, 7 B.L.R. 1-475 (1984); *Hess v. Clinchfield Coal Co*., 7 B.L.R. 1-295 (1984); *Crider v. Dean Jones Coal Co*., 6 B.L.R. 1-606 (1983); *Peabody Coal Co. v. Lowis*, 708 F.2d 266, 5 B.L.R. 2-84 (7th Cir. 1983); *see also Stevenson v. Windsor Power House Coal Co*., 6 B.L.R. 1-1315 (1984). However, the weight of the evidence, and determinations concerning credibility of medical experts and witnesses, is for the administrative law judge. *Mabe v. Bishop Coal Co*., 9 B.L.R. 1-67 (1986); *Brown v. Director, OWCP*, 7 B.L.R. 1-730 (1985); *see also Roberts v. Bethlehem Mines Corp*., 8 B.L.R. 1-211 (1985); *Henning v. Peabody Coal Co*., 7 B.L.R. 1-753 (1985); *Peabody Coal Co. v. Benefits Review Board*, 560 F.2d 797, 1 B.L.R. 2-133 (7th Cir. 1977).

As the trier-of fact, the administrative law judge has broad discretion to assess the evidence of record and determine whether a party has met its burden of proof. *Kuchwara v. Director, OWCP*, 7 B.L.R. 1-167 (1984). In considering the evidence on any particular issue, the administrative law judge must be cognizant of which party bears the burden of proof. Claimant has the general burden of establishing entitlement and the initial burden of going forward with the evidence. *See White v. Director, OWCP*, 6 B.L.R. 1-368 (1983).

Drs. Wecht, Green, Elnicki, and Abraham agreed that the miner suffered from cor pulmonale, which was the cause of the fatal cardiac arrhythmia. Drs. Oesterling and Kleinerman concluded that cor pulmonale did not exist, Drs. Tuteur and Perper did not discuss cor pulmonale in their reports, and Dr. Naeye did not discuss cor pulmonale in his report but testified in his deposition that it did not exist.[12]

Judge Leland found that Dr. Wecht's analysis was simplistic and I agree. I do not, however, take exception to his findings and measurements made during the autopsy, as other pathologists rendering opinions herein concur that he follows accepted practice and his methods of submitting slides as "excellent."[13] Therefore, while I accord Dr. Wecht's opinion little weight based on its analysis, I find that his general findings and observations on autopsy are acceptable.

Dr. Green, an eminent pathologist who has researched and published extensively in the area of pulmonary emphysema and coal dust exposure, was also in charge of the National Coal Workers' Autopsy study. Dr. Green opined that the miner had anatomic evidence of cor pulmonale, that the miner died due to cardiac arrest secondary to heart disease from cor pulmonale, which resulted from chronic lung disease. The chronic lung disease was coal workers' pneumoconiosis of mild severity, as well as moderately severe emphysema, both arising from coal dust exposure.[14] In forming his opinion, Dr. Green described a progression of

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[12] Claimant's objection to this part of Dr. Naeye's testimony was overruled, *supra*.

[13] *See* depositions of Drs. Green and Oesterling, *supra*.

[14] Regulatory amendments, effective January 19, 2001, state:
(a) For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical", pneumoconiosis and statutory, or "legal", pneumoconiosis…

pulmonary disease resulting in right-sided heart failure (cor pulmonale), based upon the gross findings in the autopsy report, autopsy slides, physical status of the miner in life, social history, status of internal organs such as the liver and spleen, and substantial and current research in the field. Dr. Green also provided extensive testimony and reports that further supported his opinions, including his reasons for rejecting the contrary opinions of the other doctors. Indeed, Dr. Green's explanation of the mechanism by which emphysema destroys lung tissue and thereby the evidence of pneumoconiosis was helpful. In addition, Dr. Green's explanation that he relied on the prosector's gross description of the lung because of the difficulty of finding emphysema on a slide alone, was also instructive. Thus, what makes Dr. Green's opinion particularly compelling is the manner in which he explained the basis for his opinions, by citing from his own and other physician's extensive research in the relevant areas and the physical findings on autopsy. I also find that Dr. Green's opinions were well-documented because he considered not just the objective evidence such as the slides and ventilatory studies, but he integrated the miner's physical condition and gross autopsy findings into his opinion. His opinion is therefore entitled to great weight.

Dr. Elnicki, board certified in internal medicine and the miner's treating physician, submitted a second report which opined that the miner died of sudden cardiac death most likely due to an arrhythmia, that the miner's pulmonary disease played a significant role in his death, and that the cause of his pulmonary disease was largely his occupational exposure to materials he encountered as a coal miner. Judge Leland initially found that Dr. Elnicki's report was entitled to less weight because he did not review as much evidence, that his credentials were not in the record, and that his opinion was not as definite as Dr. Tuteur's. Although I note that Dr. Elnicki's credentials are now in the record and that he reviewed more records, I also find that his opinion is equivocal as to the cause of death, *i.e.*, an arrhythmia. All of the physicians agree the miner experienced a fatal arrhythmia, they just differ on the cause of the arrhythmia. Therefore, I find that Dr. Elnicki's opinion is entitled to less weight.

Dr. Abraham, a board certified pathologist, also opined that the miner's coal mine employment was the source of the dust exposure in his lungs at autopsy, the cause of his pneumoconiosis, and a substantial contributing cause of his cor pulmonale and death. While Dr. Abraham provided a strong explanation of cor pulmonale, he did not discuss the objective data in relation to the miner's physical condition during his lifetime or the gross findings on autopsy, nor did he address the factor of left-sided heart failure in his report. He explained in his deposition that although he did not address the left-sided heart failure in his report, he did consider it, and he did not mention it because that factor did not address the issue of whether coal mining was a substantial factor in the miner's death. In order to understand the basis for a physician's opinion, a better reasoned opinion sets forth the factors that a physician considered and ruled out and the explanations for both in reaching his conclusion. Therefore, I find that his opinion is not well documented and is entitled to less weight.

> (2) Legal Pneumoconiosis. "Legal Pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment…
>
> (b) For purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

20 C.F.R. § 718.201.

Dr. Oesterling is board certified in anatomical and clinical pathology. Dr. Oesterling opined that the miner had no disease arising out of mine dust exposure, that the very mild dust deposition in his lungs is insufficient to have altered pulmonary function and that it did not hasten, contribute to, or cause his death. I find that Dr. Oesterling's opinion is not as well reasoned as the other pathologists', including Dr. Abraham. Dr. Oesterling opined in his reports and deposition testimony that the miner had no disease arising out of coal mine dust exposure—an opinion that differs from all the other pathologists', who each found pneumoconiosis. In addition, Dr. Oesterling's opinion did not consider the size of the right ventricle in relation to the left, focused entirely on left ventricular failure, and did not discuss the gross findings on autopsy such as clubbing of the fingers, chest diameter, or the enlarged liver and spleen. Finally, Dr. Oesterling completely rejected coal mine dust exposure as a factor in the miner's emphysema, instead attributing it to his smoking and age. Dr. Oesterling stated that "centrilobular emphysema is no more prevalent in coal miners than in the general population," a view that has been rejected by the Department of Labor. *See* 65 Fed. Reg. 79920, 79942 (Dec. 20, 2002). Therefore, I accord his opinion little weight.

Dr. Kleinerman, as noted by Judge Leland, "is a renowned expert on occupational pulmonary diseases" and is a board certified pathologist. Dr. Kleinerman opined that the autopsy slides of the miner's lungs showed a very minimal amount of coal workers' pneumoconiosis which did not cause, contribute to, or hasten his death. Dr. Kleinerman also ruled out cor pulmonale because cor pulmonale cannot be found when there is bi-ventricular hypertrophy. Dr. Kleinerman opined that the miner did not have centrilobular emphysema, which is inconsistent with the opinions of the other pathologists, including Dr. Wecht, who noted and measured the emphysematous blebs and bullae. Dr. Kleinerman's opinion that cor pulmonale cannot be ruled out in the presence of left ventricular hypertrophy is also inconsistent with the opinions of Drs. Green, Perper, and Oesterling. In addition, although Dr. Kleinerman concluded that the miner died as a result of cardiac arrhythmia that led to cardiac arrest, he does not discuss or explain a source or cause of the cardiac arrhythmia. Therefore, I find that Dr. Kleinerman's opinion is not as well reasoned as Dr. Green's and is entitled to less weight.

Dr. Naeye, a board certified pathologist who is on the faculty at the Penn State University College of Medicine, was appointed to the original committee of the American College of Pathologists to identify the components of coal workers' pneumoconiosis. Dr. Naeye concluded that the miner had minimal findings required to diagnose coal workers' pneumoconiosis, the evidence is very strong that the miner had no clinically significant disorders of his lungs, and the focal areas of fibrosis in the left ventricular wall of the heart are a plausible site from which his fatal cardiac arrhythmia could have arisen. Dr. Naeye did not address cor pulmonale or right ventricular hypertrophy in his medical report.

Dr. Naeye's deposition testimony included inconsistencies. In his deposition testimony, Dr. Naeye said he disagreed with the diagnosis of cor pulmonale because he believed that Dr. Wecht, for many years, incorrectly measured the thickness of the ventricles. Yet, Dr. Naeye stated that he was not aware of Dr. Wecht's current practice, although Dr. Wecht provided the procedure in his report. Dr. Green, however, stated that Dr. Wecht followed the current accepted practice. Dr. Naeye testified that coal mine dust exposure has only about a 5% role in emphysema, as compared to cigarette smoke, but in a 1972 JAMA article, his own research

showed a clear relationship between coal mine dust and centrilobular emphysema. Additionally, Dr. Naeye testified that he did not think there is any information available that would make it possible to determine the contribution that years of coal mine dust exposure makes to centrilobular emphysema, despite his reading and testifying about Dr. Green's deposition, which provided a citation to Dr. Green's article on the subject (which was published in the *American Journal of Epidemiology*). I also note that Dr. Naeye based his opinion on the miner having a 31-pack-year smoking history. He was unable to determine from what records he derived this figure, but this smoking history conflicts with the testimony of all the other doctors and the miner's widow. All the other evidence and the widow's testimony indicate that the miner smoked twelve to fifteen years.[15]

Dr. Naeye also did not integrate the miner's gross and physical findings, such as clubbing of the fingernails, the emphysematous blebs and bullae, and increased antero-posterior diameter of the chest. These are all indications of significant lung disease, according to Drs. Green and Wecht, yet Dr. Naeye opined that the evidence was very strong that the miner had no clinically significant disorders in his lungs. Thus, I find that Dr. Naeye's opinion is not well documented or reasoned.

Dr. Tuteur, a board certified pulmonologist, did not address cor pulmonale and opined that the miner did not have clinically significant, physiologically significant, radiographically significant, or pathologically significant coal workers' pneumoconiosis or any coal mine dust disease process, and as such, neither coal workers' pneumoconiosis nor any coal mine dust-induced disease contributed to cause his death. He further opined that the death was related to small vessel disease, myocardial ischemia, conduction abnormality, and cardiac arrest at death. Dr. Tuteur's opinion, like Dr. Oesterling's, is inconsistent from those of all the other physicians who found pneumoconiosis in this case.

Judge Leland credited Dr. Tuteur's opinion over Dr. Elnicki's in part because he reviewed more medical evidence than Dr. Elnicki. However, Dr. Tuteur's opinion was based only on the reports of Drs. Wecht and Oesterling, and Dr. Elnicki's most recent opinion was based on more medical evidence than that. Dr. Tuteur also opined that one would expect resolution of symptoms after cessation of coal dust exposure, but this was contradicted by Dr. Wecht, who testified in his deposition that the disease process of coal workers' pneumoconiosis can progress in the absence of further exposure to coal dust and the clinical signs and symptoms may worsen because the disease continues to wreak havoc with the pulmonary system and, in some instances, the cardiovascular system. In addition, the regulations and the relevant case law provide that pneumoconiosis is a latent and progressive disease that may first become detectable only after the cessation of coal mine dust exposure. 20 C.F.R. § 718.201(c). Therefore, I accord his opinion little weight and less weight than Dr. Elnicki's.

Dr. Perper, a pathologist,[16] concluded that the miner had minimal coal workers' pneumoconiosis as a result of exposure to mixed coal dust, that the coal workers'

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[15] This figure was also posed to Dr. Green in his depostion and employer's counsel stated that it came from Dr. Elnicki's report. I note, however, that Dr. Elnicki wrote that the miner smoked twelve years.

[16] I took judicial notice of that fact that he is a forensic pathologist and an attorney, *supra* at n.7.

pneumoconiosis was too mild to be a substantial, contributory, or hastening factor in the miner's death and that death was due to cardiac dysrhythmia on the background of cardiomyopathy with cardiomegaly and focal myocardial fibrosis. Dr. Perper's opinion, in its initial query on pneumoconiosis causation, stated that the miner had only twelve years of coal mine employment as opposed to 34. While Dr. Perper noted that the heart was enlarged, he did not discuss either right or left sided hypertrophy, but instead opined that the miner's death was caused by hypertrophic cardiomyopathy. According to Dr. Green, this condition is very rare and he has never seen it in his career. In addition, Dr. Green stated that hypertrophic cardiomyopathy causes a specific thickening of the heart muscle near the left ventricle that would be very apparent on autopsy. This was not reported by Dr. Wecht on autopsy. Dr. Perper's report does not integrate the physical and gross autopsy findings. Therefore, I find that it is not as well reasoned as Dr. Green's and is entitled to less weight.

In terms of the qualifications of the pathologists rendering opinions, I find that Drs. Green and Kleinerman are the most qualified with respect to both experience and published research. Dr. Perper's curriculum vitae is not in the record. Dr. Naeye also has impressive credentials; however, his testimony indicated that he is not as up to date with the current and relevant research and literature, nor has he researched or published in this area in recent years. While Dr. Oesterling has a great deal of experience, unlike the others he is not on the faculty of a university and he has not published research in this area.

I find that not only is Dr. Green, along with Dr. Kleinerman, the most qualified physician, but also that his medical reports and testimony were better documented and reasoned than those of any of the contrary physicians, including Dr. Kleinerman. I find that Dr. Green's opinion—that the miner died due to cardiac arrest secondary to cor pulmonale, which resulted from chronic lung disease arising from his coal mine employment—best synthesizes all of the medical evidence of record. Weighing all of the evidence together, I find that the Claimant has met her burden of showing that the miner's pneumoconiosis was a substantially contributing cause or factor leading to his death under the standards imposed by § 718.205(c).

Payment of Deposition Expenses

At the hearing, Claimant requested an order that Employer pay the cost of the deposition of Claimant's expert witness, Dr. Green. A discussion ensued as to applicability of the "old" or "new" regulations to this case. I directed the parties to file briefs on the applicability of the regulations to this issue and instructed Claimant's counsel to file a specific motion regarding the payment of Dr. Green's deposition costs. Employer's attorney submitted its response to Claimant's motion[17] and conceded that it is responsible for payment of the costs of Dr. Green's deposition. Accordingly, Employer is hereby ordered to pay the expenses associated with Dr. Green's deposition.

[17] The response is dated August 21, 2003 and is entitled Shannopin's Response to Wiltrout's Motion for Payment of Deposition Expenses and Brief on Applicability of Regulations. Employer conceded its responsibility for payment of Dr. Green's deposition costs at p.1.

Entitlement to Benefits

Because Claimant established all of the requisite elements of entitlement, I find Claimant is entitled to benefits under the Act.

Date of Onset

The regulations provide that in modification cases based on determination of a mistake in fact, the onset date is determined by § 715.503(c). *See* 20 C.F.R. § 725.503(d)(2001). This section provides that in a survivor's claim, benefits are payable beginning with the month of the miner's death, or January 1, 1974, whichever is later. The miner died on June 4, 1997. Accordingly, the date of onset is June 1, 1997.

Attorney's Fees

No award of attorney's fees for services to Claimant is made herein since no application has been received. Thirty days are hereby allowed to Claimant's counsel for the submission of such application. His attention is directed to 20 C.F.R. §§ 725.365 and 725.366 of the Regulations. A service sheet showing that service has been made upon all parties, including the Claimant, must accompany the application. Parties have ten days following receipt of such application within to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

The claim of EDITH K. WILTROUT for black lung benefits under the Act is hereby GRANTED, and

It is hereby ORDERED that SHANNOPIN MINING COMPANY, the Responsible Operator, shall pay to the claimant, EDITH K. WILTROUT, all benefits to which she is entitled under the Act, commencing as of June 1, 1997 and shall pay the expenses associated with Dr. Green's deposition.

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MICHAEL P. LESNIAK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS. Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this Decision and Order was filed in the Office of the District Director, by filing a notice of appeal with the *Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601*. A copy of a notice of appeal must also be served on Donald S. Shire, Esq., Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution